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Resistance Training Promotes Reduction in Blood Pressure and Increase Plasma Adiponectin of Hypertensive Elderly Patients

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Abstract

Introduction: Hypertension is the most common disease in industrialized countries, and it is responsible for the major risk factor for developing cardiovascular diseases. Aging increases the prevalence of hypertension and the loss of muscle mass. The aim of the present study was to evaluate the effect of resistance training on blood pressure and inflammatory markers in hypertensive elderly.

Methods: One hundred thirty-five hypertensive elderly were distributed into two groups: training (TG, n=86), and control (CG, n=49). TG underwent 16 weeks of resistance training with three sets of 12 repetitions in eight exercises (50% of one maximal repetition) and the CG remained sedentary. Twenty-four-hour ambulatory blood pressure was recorded.

Results: Resistance training increased muscle strength (21.10 ± 0.71 vs. 25.76 ± 0.80 kg [p<0.01] chest press; 9.93 ± 0.29 vs. 14.41 ± 0.45 kg [p<0.01] knee extension), as well as reduced systolic blood pressure (126.79 ± 1.29 vs. 124.32 ± 1.37 mmHg [p<0.05] daytime; 125.11 ± 1.28 vs. 122.50 ± 1.38 mmHg [p<0.05] 24-h period) in TG. In addition, resistance training increased the circulating levels of adiponectin (4.61 ± 0.24 vs. 5.32 ± 0.25 pg/mL [p<0.01]) and reduced the circulating levels of ICAM-1 (334.38 ± 10.89 vs. 293.05 ± 9.78 pg/mL [p<0.01]).

Conclusion: Our results suggest that resistance training is an important tool to reduce blood pressure and improve muscle strength in hypertensive elderly. Besides that, resistance training increase circulating levels of adiponectin, and reduce the levels of plasma ICAM-1 in hypertensive elderly.

Keywords

Hypertension; Elderly; Resistance training; Inflammatory status

Abbreviations:

SAH: Systemic Arterial Hypertension; TNF-α: Tumor Necrosis Factor – Alpha; CRP: C-Reactive Protein; TG: Training Group; CG: Control Group; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; IPAQ-6: International Physical Activity Questionnaire; ABPM: Ambulatory Blood Pressure Monitoring; MR: Maximal Repetition; HDL-c: High Density Cholesterol; LDL-c: Low Density Lipoprotein Cholesterol; IL-6: Interleukin-6; PAI-1: Plasminogen Activator Inhibitor Type 1; VCAM-1: Vascular Cell Adhesion Molecule-1; ICAM-1: Intracellular Adhesion Molecule-1

Introduction

Systemic arterial hypertension (SAH) is the most common disease in industrialized and developing countries [1]. With aging, the prevalence of systemic arterial hypertension increases, affecting 58% of women and 53% of men ≥ 65 years old in the USA [2].

SAH is the major factor in the development of cardiovascular disease, such as left ventricular hypertrophy and endothelial dysfunction [3], thus increasing the risk of cardiovascular-related deaths.

Some authors have correlated an increase in blood pressure levels with an increase in circulating levels of pro-inflammatory cytokines [4], such as tumor necrosis factor alpha (TNF-α) and C-reactive protein (CRP) [5], although the underlying mechanisms are unclear. Besides that, hypertensive patients exhibit impairment in anti-inflammatory cytokines [4,6,7] such as plasma adiponectin when compared with normotensive individuals [6,8]. Furthermore, systemic arterial hypertension stimulates the production of adhesion molecules [9].

Aging enhances the risk for SAH [10] and promotes loss of muscle mass [11]. Such loss of strength can decrease independence, and is the major cause of falls and injuries in this age group [12]. Nevertheless, the elderly maintain muscular plasticity, which is a favorable feature in the recovery of muscle mass and muscle strength when the elderly engage in resistance training [13].

The importance of aerobic exercise is well known as a tool in the non-pharmacologic treatment of systemic arterial hypertension, thus data concerning blood pressure and pro and anti-inflammatory cytokines after periods of resistance training in hypertensive elderly are limited and controversial [14].

The purpose of this study was to evaluate the effect of 16 weeks of resistance training on blood pressure, inflammatory markers, lipid profile and plasma glucose of hypertensive elderlies.

Methods

Experimental group

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One hundred forty two sedentary hypertensive elderly old without limitations on physical activity were distributed into training (TG, n=87) or control (CG, n=55) groups, TG was filled out with invited patients from a particular clinic, and CG with volunteers recruited by announcement for the community. However, seven volunteers dropout during the study protocol, one from TG and other six from CG (TG, n=86; CG, n=49). Inclusion criteria were age between 60 and 80 years (68.04 ± 0.52 years of age (Table 1)), subjects were classified as hypertensive if prescribed any class of anti-hypertensive drugs or had three measurements on non-consecutive days of systolic blood pressure (SBP) readings >139 mmHg and/or diastolic blood pressure (DBP) readings >89 mmHg. Volunteers were required to be sedentary at least 6 months prior to the beginning of the study. This criteria was validated by applying the short form of the International Physical Activity Questionnaire (IPAQ-6). Exclusion criteria were as follows: change in drug treatment during the protocol; and orthopedic conditions or other conditions that prevent the subject from performing any exercise of the resistance training protocol. Medical histories and clinical evaluations were used to exclude any subject with other cardiovascular, renal, or pulmonary disorders and chronic physical or psychological disorders. All volunteers read a detailed description of the protocol and provided written informed consent. The Ethics Committee of Federal University of São Paulo, SP, Brazil, approved all procedures (0451/11).

Ambulatory blood pressure monitoring

Blood pressure was measured using ambulatory blood pressure monitoring ([ABPM], Dyna-MAPA, Cardios®; [São Paulo, SP, Brazil]) before and after the experimental period at 9 am on days that the volunteers did not perform any physical activity. The volunteers were asked to record the hours of sleep, the time of awakening, and the schedule of medications were taken. The equipment recorded blood pressure every 15min during the daytime (according to volunteer report) and every 30 min during the nighttime (according to volunteer report). Blood pressure recordings were assessed in three periods for systolic and diastolic pressures and heart rate: 24-h period; daytime and nighttime according to sleeping hour related in the diary.

Resistance exercise training protocol

The resistance training protocol was registered on Clinical Trials (www.clinicaltrials.gov) under protocol number NCT01963507. The TG underwent 16 weeks of resistance training, which was performed three times per week on non-consecutive days at 9 am. Protocol training consisted of3sets of 12 repetitions in 8 exercises (Nakagym® and Biodelta®; [São Paulo, SP, Brazil]), with 90s of rest between sets and 2 min of rest between exercises. Each training session lasted 60 min. The CG remained sedentary during the same time period. Before six sessions of habituation, both groups performed maximal repetition (MR) tests in all exercises proposed in the training protocol, following the protocol proposed by Fleck and Kraemer [15]. The TG repeated the MR protocol every 4 weeks to maintain the relative workload on the protocol training in 50% of 1MR. During the last week of the protocol, both groups repeated the MR protocol. The room temperature was maintained at 24°C. Volunteers were asked not to perform any physical activity not specified in the experimental protocol, but to maintain their daily activities, as well as to maintain their food intake. Subjects of the CG were asked to maintain their normal daily activities, as well as, food intake.

Blood content analysis

Fasting venous blood was collected to perform biochemical analyses. The collected blood was centrifuged for 15 min at 3500× *g*. Serum was collected and frozen at −80°C. Cytokines were evaluated by ELISA kits from R&D Systems ([Minneapolis, MN, USA]): CRP (SCRP00); adiponectin (DY1065); TNF-α (SSTA00D); interleukin-6 (IL-6;SS600B); plasminogen activator inhibitor type 1 (PAI-1;DY1786); vascular cell adhesion molecule-1 (VCAM-1;DY809); and intracellular adhesion molecule-1 (ICAM-1;DY720). The lipid profile was assessed by colorimetric assay with kits from Labtest (Lagoa Santa, MG, Brazil): fasting glycemia; total cholesterol; High Density cholesterol (HDL-c); and triacylglycerol. Low Density Lipoprotein cholesterol (LDL-c) was calculated using the Friedwald equation [16].

Statistical analysis

Since our studied group was not randomly assigned, percentage of genders and some variables were significant different between groups prior to the study, we performed the analysis of covariance (ANCOVA Two-Way) in order to reduce statistical bias. We employed the value pre intervention of the variables as covariate. The significance level was set at $p \leq 0.05$. Data are presented as the mean ± standard error of the mean. Software Statistica 10.0 was used to perform statistical analyses.

Results

Subject characteristics

The anthropometric data did not change after experimental protocol in either group, however, the CG had lower weight compared to TG in both periods of the study (Table 1). TG showed an increase in muscle strength in all of the performed exercises, and the increases were greater than observed in the CG (Table 2).

Ambulatory blood pressure monitoring

The TG had a lower blood pressure during the daytime (systolic and diastolic blood pressure) compared to the CG before the experimental protocol, however, resistance training further reduced the systolic blood pressure (126.79 ± 11.29 vs. 124.32 ± 1.37 mmHg [$p<0.05$]; Table 3). During the nighttime, systolic and diastolic blood pressure were reduced in the TG compared to the CG before experimental protocol. In this period, both groups did not alter blood pressure after the experimental protocol (Table 4).

During 24h period, TG had lower blood pressure before the experimental protocol compared to the CG. Resistance training reduced systolic blood pressure after the experimental protocol in TG (125.11 ± 1.28 vs. 122.50 ± 1.38 mmHg [$p<0.05$] Table 5). In contrast, both groups increased the heart rate after the experimental protocol (69.35 ± 1.05 vs. 74.51 ± 1.05 bpm [$p<0.001$]; 65.13 ± 1.75 vs 72.78 ± 2.25 bpm [$p<0.001$]; TG and CG, respectively; Table 5).

Biochemical and Inflammatory markers analysis

The lipid profile was similar in both groups before the experimental protocol, and no changes were observed in the TG, except for triacylglycerol, that was lower in CG prior the study, and had a reduction in the circulating levels after the experimental protocol (208.32 ± 13.33 vs. 166.21 ± 11.12 mg/dL [$p<0.001$]; Table 6). Fasting glycemia was similar between groups before the experimental protocol and remain unaltered after experimental protocol in both groups.

At the beginning of the study, the circulating levels of VCAM-1 were reduced in the TG compared to the CG; however, the CG had reduced circulating levels of VCAM-1 after the experimental protocol (547.93 ± 23.84 vs. 442.25 ± 26.40 ng/mL [$p<0.01$]; Table 6). The TG

Anthropometric data Pre and Post experimental protocol				
	TG		CG	
	Pre	Post	Pre	Post
n	86	-	49	-
Women	65	-	26	-
Age (years)	67.45 ± 0.63	-	69.04 ± 0.94	-
Height (cm)	159.3 ± 0.01	-	163.30 ± 0.01	-
Weight (Kg)	74.55 ± 1.47#	74.02 ± 1.45#	71.79 ± 1.82	71.82 ± 1.78
BMI (Kg/m²)	29.36 ± 0.54#	29.16 ± 0.54#	26.87 ± 0.58	26.88 ± 0.57
Pharmacologial treatment	64	-	39	-
RAAS Drug	58	-	35	-
* p<0.05 Pre vs Post, # p<0.05 TG vs CG. BMI = Body Mass Index; TG= Training Group; CG= Control Group; Cm = Centimeters; Kg= Kilograms; RAAS=Renin-angiotensin-aldosteron-system				

Table 1: Anthropometric data Pre and Post experimental protocol

Maximal repetition tests Pre and Post experimental protocol				
	TG		CG	
	Pre	Post	Pre	Post
Chest Press (Kg)	21.10 ± 0.71	25.76 ± 0.80*#	21.31 ± 0.93	22.63 ± 0.96
Knee Extension (Kg)	9.93 ± 0.30	14.41 ± 0.45* #	9.02 ± 0.33	10.93 ± 0.75*
Seated Row(Kg)	22.94 ± 0.69	27.55 ± 0.73* #	23.55 ± 0.86	24.19 ± 0.78
Leg Curl (Kg)	7.77 ± 0.19	11.64 ± 0.33* #	8.10 ± 0.47	9.44 ± 0.67*
Triceps Extension (Kg)	19.54 ± 0.44	24.81 ± 0.62* #	19.25 ± 0.81	19.25 ± 1.03*
Seated Hip Adduction (Kg)	34.07 ± 0.38	40.09 ± 0.61* #	32.66 ± 0.74	34.46 ± 1.00*
Biceps Curl (Kg)	5.52 ± 0.15	7.87 ± 0.20* #	6.21 ± 0.26	6.76 ± 0.26*
Leg Calf Raise (Kg)	12.50 ± 0.28	16.75 ± 0.48* #	11.38 ± 0.65	12.04 ± 0.77
* p<0.05 Pre vs Post, # p<0.05 TG vs CG. TG= Training Group; CG= Control Group; Kg= Kilograms				

Table 2: Maximal repetition tests Pre and Post experimental protocol

Blood Pressure during Daytime				
	TG		CG	
	Pre	Post	Pre	Post
SBP (mmHg)	126.79 ± 1.29#	124.32 ± 1.37 *#	134.67 ± 2.24	133.50 ± 2.30
DBP (mmHg)	72.74 ± 0.87#	72.08 ± 1.01 #	78.97 ± 1.26	79.00 ± 1.31
HR (BPM)	77.53 ± 1.08	76.94 ± 1.08	75.28 ± 2.02	76.65 ± 2.26
* p<0,05 Pre vs Post, # p<0,05 TG vs CG. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate				

Table 3: Blood Pressure during Daytime

Blood Pressure during Nighttime				
	TG		CG	
	Pre	Post	Pre	Post
SBP (mmHg)	120.69 ± 1.48#	118.02 ± 1.59#	127.43 ± 2.46	124.50 ± 2.81
DBP (mmHg)	66.47 ± 0.99#	65.01 ± 1.10#	71.41 ± 1.37	72.89 ± 1.83
HR (BPM)	69.35 ± 1.05#	68.54 ± 1.07	65.13 ± 1.75	67.19 ± 2.45
* p<0,05 Pre vs Post, # p<0,05 TG vs CG. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate				

Table 4: Blood Pressure during Nighttime

had an increase in adiponectin levels (4.61 ± 0.24 vs. 5.32 ± 0.25 ng/mL [p<0.001]; Table 6) and a decrease in the circulating levels of ICAM-1 (334.38 ± 10.89 vs. 293.05 ± 9.78 ng/mL [p<0.001]; Table 3). The other markers of inflammation analyzed, C reactive protein, TNF-α and PAI-1 were equal between groups in the beginning of the study, and the experimental protocol did not alter their circulating levels. However, IL-6 was higher in TG at the beginning of the study and did not change after experimental protocol in both groups remaining higher in TG.

Discussion

The main results achieved in hypertensive elderly patients included a significant increase in muscle strength, a reduction in daytime and 24-h systolic blood pressure, and an improvement in inflammatory status after 16 weeks of resistance training.

The guidelines for resistance exercise in individuals with and without cardiovascular disease promulgated by the American Heart Association [17] point to modifications in body composition in those who engage in a program of resistance training >12 weeks in length. Such alterations refer to enlargement in muscle mass. We did not find alterations in anthropometric data in either group, however, we found improvements in muscle strength mainly in TG ($p< 0.01$). Our data are similar to the findings of Takarada et al. [18], which observed an improvement in muscle strength in elderly females but did not observe changes in the cross-sectional area of knee extensors and flexors.

Population studies conducted with normotensive patients showed that resistance training produces reductions in clinical systolic and diastolic blood pressure [14], however, studies conducted with hypertensive patients are limited. In a meta-analysis, Cornelissen and Fagard [19] analyzed 33 studies, thus, only 5 were conducted with hypertensive subjects. The results of this meta-analysis showed reductions in blood pressure among individual with optimal blood pressure or prehypertensive, thus hypertensive subjects did not experienced reductions in blood pressure. In the present study, we found a significant reduction in the systolic blood pressure during the daytime in TG ($p<0.05$). Similar results were reported by Melo et al. [20] after an acute exercise session of resistance training. The group studied consisted of women receiving captopril, whereas our group was heterogeneous in gender and pharmacologic treatment. In

Blood Pressure during 24h period				
	TG		CG	
	Pre	Post	Pre	Post
SBP (mmHg)	125.11 ± 1.28#	122.50 ± 1.38*	132.65 ± 2.17	129.69 ± 2.57
DBP (mmHg)	71.09 ± 0.85#	70.10 ± 1.00#	76.89 ± 1.21	78.06 ± 1.58
HR (BPM)	69.35 ± 1.05#	74.51 ± 1.05*	65.13 ± 1.75	72.78 ± 2.25*
* p<0,05 Pre vs Post, # p<0,05 TG vs CG. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate				

Table 5: Blood Pressure during 24h period

Lipid profile, inflammatory status Pre and Post experimental protocol				
	TG		CG	
	Pre	Post	Pre	Post
Total Cholesterol (mg/dL)	199.10 ± 7.30	198.08 ± 8.20	202.47 ± 7.63	192.09 ± 8.72
LDL-c (mg/dL)	110.53 ± 8.39	114.24 ± 9.20	101.76 ± 8.33	121.50 ± 7.92
HDL-c (mg/dL)	63.15 ± 3.80	66.70 ± 4.08	74.75 ± 7.11	65.76 ± 6.39
Triacylglycerol (mg/dL)	145.15 ± 5.30#	138.20 ± 5.59#	208.32 ± 13.33	166.21 ± 11.12*
Fasting Glycemia (mg/dL)	120.72 ± 3.12	123.42 ± 3.22	121.13 ± 5.27	116.20 ± 5.50
CRP (ng/mL)	24.81 ± 1.87	25.51 ± 1.79	21.95 ± 2.60	21.94 ± 2.59
TNF-α (pg/mL)	6.64 ± 1.03	7.02 ± 1.60	4.75 ± 0.54	9.00 ± 2.96
IL-6 (pg/mL)	8.20 ± 2.01#	7.67 ± 2.00#	2.75 ± 1.04	2.75 ± 1.05
Adiponectin (pg/mL)	4.61 ± 0.24	5.32 ± 0.25*	5.16 ± 0.34	5.01 ± 0.16
VCAM-1 (pg/mL)	429.36 ± 11.53#	399.30 ± 14.46	547.93 ± 23.84	442.25 ± 26.40*
ICAM-1 (pg/mL)	334.38 ± 10.89	293.05 ± 9.78*	309.69 ± 20.42	301.67 ± 18.22
PAI-1 (pg/mL)	6610.15 ± 523.59	6112.67 ± 490.57	8020.01 ± 608.93	8119.09 ± 643.48
* p<0.05 Pre vs Post, # p<0.05 TG vs CG. LDL-c = Low Density Lipoprotein cholesterol, HDL-c = High Density cholesterol, CRP = C Reactive Protein, TNF-α = Tumor Necrosis Factor Alpha, IL-6 = Interleukine-6, VCAM-1 = Vascular Cell Adhesion Molecule-1, ICAM-1 Intracelular Adhesion Molecule-1, PAI-1 = Plasminogen Activator Inhibitor-1.				

Table 6: Lipid profile, inflammatory status Pre and Post experimental protocol

addition, the training protocol in that study involved 20 repetitions and 40% of 1MR. Another study [21] evaluated the influence of one single bout of resistance exercise conducted with hypertensive subjects using a protocol similar to ours, thus significant reductions in systolic and diastolic blood pressure were observed and those reductions were maintained up to 1 hour after the exercise session.

During the nighttime blood pressure in TG did not changed, as also occurred in the CG. Studies conducted with hypertensive subjects [20,21] who underwent one single session of resistance training did not demonstrate alterations in blood pressure during the nighttime.

Different of studies conducted with hypertensive subjects that did not reveal alterations in blood pressure in the trained groups [20,21] our results support the hypotensive effects of resistance training, since during the 24-h period, resistance training promoted reduction in systolic blood pressure ($p<0.05$). However, diastolic and mean blood pressure did not change after the experimental protocol in both groups.

Overall, the present study observed small but significant reductions in systolic blood pressure elicited by resistance training. It is important to note that Whelton et al. [22] demonstrated that reductions in systolic blood pressure of 2 mmHg can reduce the risk of stroke by 6%, chronic heart disease by 4%, and overall mortality by 3%.

Resistance training was not effective in reducing fasting glycemia ($p=0.2$), total cholesterol ($p=0.6$) and fractions ($p=0.5$); nevertheless, a meta-analysis [23] evaluated seven studies that assessed the effects of resistance training on fasting glycemia of subjects with type 2 diabetes, and similar results were obtained (i.e., a slight reduction in fasting glycemia in the studied groups). In another study with sedentary elderly, Davidson et al. [24] did not find alterations in fasting glycemia, as in the present study.

The adiponectin level has an inverse correlation with systemic arterial hypertension [6-8]. Hypoadiponectinemia has been suggested to be a risk factor for developing systemic arterial hypertension [7]. The main mechanism that explains this correlation involves the phosphorylation of adiponectin receptors (AdipoR1 and AdipoR2), which increases the expression of nitric oxide synthase[8]. The present study showed an improvement in the circulating levels of adiponectin ($p<0.01$), even with maintenance of body weight, highlighting a feasible cardioprotective factor elicited by resistance training. Our data are opposed to the findings in some studies that did not observe alterations in adiponectinemia after the resistance and aerobic training periods [25,26]. However, Fatouros et al. [27] using a protocol with three different intensities of exercise, found improvement in circulating levels of adiponectin in groups who trained with moderate and high intensity, as well as Brooks et al. [28], which assessed elderly subjects with type 2 diabetes and found increases in adiponectinemia after 16 weeks of moderate to high intensity resistance training. Those findings substantiate that improvement in adiponectinemia is intensity-dependent, however, our study, find that even with low intensity, resistance training can increase plasma adiponectin in hypertensive elderly.

Some studies have proposed that resistance training does not influence cell adhesion molecules (VCAM-1 and ICAM-1) [29,30], however, due to an augmentation in catecholamines during resistance training, especially epinephrine, a redistribution of adhesion molecules in the endothelium could occur, since neutrophils and lymphocytes have receptors to catecholamines, and catecholamines elicits expression of specific adhesion molecules and changes in its cytoskeletal

organizations, then altering its adhesion to endothelial walls. A study conducted with young men who underwent a resistance training period did not show changes in circulating levels of cell adhesion molecules [31]. Our results are in contrast to those findings because the TG showed significant reductions in serum ICAM-1 ($p<0.01$), and interestingly CG showed reductions in serum VCAM-1 ($p<0.01$).

Wang et al. [8] correlated improvements in adiponectinemia with an increase in nitric oxide bioavailability. Nitric oxide is a mediator of production of cell adhesion molecules, once our study find augmentations in adiponectinemia and reductions in blood pressure, this fact can be correlated to the reductions observed in adhesion molecule cells and in the blood pressure levels, even we did not measured nitric oxide bioavailability.

The influence of resistance training in inflammation remains controversial [32]. Our data were similar to findings of other studies [30,33] that did not find changes in circulating levels of IL-6. Reductions in the circulating level of CRP have been shown [30,33], however, our data did not show reductions after the training period ($p=0.6$), neither in circulating levels of TNF-α ($p=0.1$). Others studies have also reported no changes in circulating levels of TNF-α [33,34]. PAI-1 is closely linked to hypertension and the levels of nitric oxide are impaired in hypertensive subjects, and plasma and endothelial levels of PAI-1 are increased [35]. Data involving physical exercise and PAI-1 are limited, however, DeSouza et al. [36] reported a reduction in PAI-1 after a single session of aerobic exercise. No alterations in PAI-1 were shown in the present study ($p=0.1$).

Briefly, resistance training in hypertensive elderly subjects improved muscle strength, reduced daytime and 24-h systolic blood pressure and ICAM-1 levels, and improved adiponectin levels; however, the mechanisms by which resistance training produced these results remain unclear.

In conclusion, the results obtained in the present study allow us to assert that resistance training promotes a reduction in blood pressure and an improvement in muscle strength. Resistance training increased circulating levels of adiponectin, and reduced the levels of plasma ICAM-1.

Study limitations

The most important limitations of our study were that the study group was not randomized and we did not limit the class and dosage of medications the volunteers were using.

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Disclosure

All authors takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation. The authors declare no conflicts of interest.

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